Transfers and transitions: Parent-offspring conflict, genomic imprinting, and the evolution of human life history

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Human offspring are weaned earlier than the offspring of other great apes but take longer to reach nutritional independence. An analysis of human disorders of imprinted genes suggests genes of paternal origin, expressed in infants, have been selected to favor more intense suckling than genes of maternal origin. The same analysis suggests that genes of maternal origin may favor slower childhood growth but earlier sexual maturation. These observations are consistent with a hypothesis in which slow maturation was an adaptation of offspring that reduced maternal fitness, whereas early weaning was an adaptation of mothers that reduced the fitness of individual offspring.

Beckwith–Wiedemann syndrome | genomic imprinting | Prader–Willi syndrome | weaning

Ethnographic data suggest our ancestors consumed more food than they gathered until early adulthood and gathered more food than they consumed thereafter (1). Thus, hominin life history involved a transfer of resources from older producers to younger consumers. Lee modeled the consequences of these transfers for the evolution of age-specific mortality (2, 3). He found that transfers from older to younger individuals mitigate the force of selection against early deaths, because the death of a dependent youngster frees food for other group members, but intensify selection against late-life mortality, because the death of a productive elder reduces food for survivors.

Lee's model assumed consumers and producers were genetically identical, except for new mutations (2). It was as if older producers could provision their younger selves. In sexual life cycles, however, resources are transferred between individuals who may share some, but not all, of their genes (4). If multiple donors transfer resources to multiple recipients, then each donor favors the distribution of resources that maximizes her inclusive fitness, but each recipient favors the distribution that maximizes his inclusive fitness. Individual consumers are predicted to take a larger share of production (if given the opportunity) than the quantity favored by donors. The paradigm of such conflict is the allocation of maternal investment among offspring. If a mother distributes resources in a manner that maximizes her fitness, then each offspring will favor a reallocation from sibs to itself (5). Genes that are expressed differently when inherited via ova than via sperm are predicted to mediate kin conflicts (6, 7). Therefore, the phenotypic effects of such imprinted genes will provide important clues about how transfers among kin have shaped human life history.

Modeling Transfers Among Kin

Patterns of resource transfers within groups and gene transfers between groups are variable among modern human populations and were presumably variable among ancestral populations. Simplification of this complexity is necessary to gain theoretical insight into how resource and gene transfers interact. I will consider a simple gene-transfer model in which females move to new groups when they switch from being net consumers to net producers whereas males remain in their natal group, and an equally simple resource-transfer model in which females put food into household pots, from which their own offspring feed, but men put food into a communal pot, from which all offspring feed. These models will be combined with a model of childhood consumption that identifies conflicts between genes of maternal and paternal origin. These verbal models are deliberately abstract because it is my belief that progress in understanding the action of natural selection in complex human social groups will be advanced by first understanding how selection acts in simpler systems.

Recent human populations exhibit a flexibility of social structures that is not captured in these models: males move to live with their wives' families; both sexes have multiple sexual partners; relationships dissolve; groups split or fuse; related individuals emigrate together; and resources, and genes, are exchanged between groups. Moreover, genes repeatedly leave and reenter local groups as a consequence of regular intermarriage between clans, creating larger regional groups bound together by interlocking kinship, and patterns of relatedness within groups are influenced by demographic stochasticity. Thus, my models assume patterns of gene and resource transfers that are undoubtedly wrong in details, and perhaps wrong in fundamentals. Nevertheless, I hope these models will identify key issues, both empirical and theoretical, that must be addressed by more-realistic analyses, and will provide a baseline against which the effects of departures from my idealized assumptions can be assessed.

One assumption of the models is particularly contentious. I assume mobile females and sedentary males. Patrilocality has been claimed to be the predominant mode of social organization among recent hunter-gatherers (8) but this claim has been strongly disputed (9, 10). My models are not intended to resolve this debate, but rather to explore the theoretical consequences of female-biased dispersal, in part, because I considered male-biased dispersal in ref. 11. Ethnographic data show that both forms of dispersal occur among recent humans and that females often maintain social ties with their natal group after dispersal. I believe that the relative mobility of the sexes in the past is a key unresolved issue for understanding the evolution of human life history.

A challenge for future models will be incorporating variability among social groups. Natural selection deals with variability in 2 contrasting ways that necessitate different modeling approaches. The first is to average across circumstances: strategies evolve that are adaptive on average even though they are maladaptive in some situations. The second is to evolve contingent strategies that enable individuals to respond in different ways to different circumstances. The first process produces a

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Table 1. Human imprinted disorders discussed in text

Human syndrome	Some (epi)genetic causes	Ref(s).
Beckwith–Wiedemann syndrome (BWS)	Excess expression of paternally-expressed *IGF2* Inactivation of maternally-expressed *CDKN1C*	23,57
Silver–Russell syndrome (SRS)	Maternal methylation pattern of paternal 11p15.5 Maternal uniparental disomy 7	23,58
Prader–Willi syndrome (PWS)	Deletion of paternal 15q11–13 Maternal uniparental disomy 15	35,59
Temple syndrome (TS)	Deletion of paternal 14q32 Maternal uniparental disomy 14	49

general-purpose phenotype and the second a repertoire of context-specific adaptations.

Gene-Transfer Model. A young woman enters a group where she lacks relatives and accumulates kin by reproduction. At first her kin are restricted to her own offspring but later include grandoffspring, as her sons mature and reproduce with younger unrelated women. By contrast, a young man remains within his natal group and obtains one or more wives from neighboring groups. The dependent kin of his group consist of his own offspring, his younger sibs, and a mixture of patrilineal nieces, nephews, and cousins. As he ages, the composition of reproductive males in his group gradually shifts from uncles and brothers to sons and nephews. At the same time, the population of dependent young shifts to grandchildren and progressively more distant patrilineal kin.

In this scenario, the productive older individuals of a group consist of unrelated females and related males. Thus, the group's genetic cohesion is maintained via patrilineal descent, including descent from shared paternal grandmothers, while the genetic fissures separate matrilines. Genetic cohesiveness declines as new females enter the group and establish new matrilines. Eventually, the group may become unstable and split, possibly into separate matrilines of older females.

Resource-Transfer Model. In the gene-transfer model, a young wife's only kin among the younger consumers of her group are her own offspring. From her genetic perspective, these are the rightful focus of her (and her husband's) provisioning. By contrast, her husband is one among a group of related males and has a genetic interest in their offspring as well as his own. Therefore, he gains more inclusive fitness from contributions to a common pot than does his wife, and has less incentive to hoard food for his own household.

For purposes of abstraction, I will assume men put food into a communal pot from which all juveniles feed whereas women put food into a household pot from which their own offspring feed. Each additional male hunter enhances the amount of resources put into the communal pot (and spreads the risks from hunting; 12) whereas each additional female gatherer competes for local resources with other households. Mothers increase their fecundity if they can shift some of the burden of childrearing from the household to the communal pot. To mix metaphors, the communal pot is a commons on which the offspring of different households graze and, if unregulated, is expected to be overstocked (3, 13). Fathers favor longer interbirth intervals (lower stocking rates) than mothers because of their greater relatedness to offspring of other households. (This prediction is sensitive to relaxation of the assumptions of stable monogamy and patrilocal residence).

Consumption Model. In the above model, offspring have 3 sources of food: items collected by their mother and contributed to the household pot, items contributed to the communal pot by adult males, and items collected by the child itself. Food taken from the household pot reduces food available to sibs whereas food taken from the communal pot reduces food available to offspring of all households. Food that an offspring forages for itself reduces demands on the household and/or the communal pot. Given a choice between taking an equivalent item from the household pot or communal pot, a child would generally prefer the communal pot because the personal benefit is the same but the cost is spread across a larger group of less-related kin.

Conflicts over resource consumption can exist within the genomes of children between genes of maternal and paternal origin. Suppose that faster feeding from a pot results in the guzzler obtaining more food (individual benefit) at the cost of greater spillage (group cost). The consumers from each household pot comprise a smaller group of maternally-derived alleles than paternally-derived alleles (because some consumers are maternal half-sibs with different fathers) whereas the consumers from the communal pot comprise a smaller group of paternally-derived alleles than maternally-derived alleles (because consumers are the progeny of related fathers but unrelated mothers). Therefore, paternally-expressed genes are predicted to promote faster eating and greater spillage from the household pot, whereas maternally-expressed genes are predicted to promote faster eating and greater spillage from the communal pot (14). If younger children are fed from the household pot but older children are fed from the communal pot, then maternally-derived alleles of children would favor graduation to the communal pot at a younger age than the age favored by paternally-derived alleles.

As a consequence of asymmetries of patrilineal and matrilineal relatedness within social groups, imprinted genes are predicted to influence how much a child consumes and at whose expense, and to accelerate or retard transitions between life-history stages.

Ontogeny of Resource Transfers

Human development is associated with a series of transitions that influence the pattern of resource transfers. After ovulation, the early embryo subsists on tubal and uterine secretions, and reserves deposited in the oocyte. After implantation, the offspring gains direct access to maternal blood via a hemochorial placenta. Parturition is marked by the abrupt loss of the placental haustorium and its replacement by suckling at the breast. Weaning is a more or less gradual process by which milk is first supplemented, and then replaced, by other foods. Adrenarche is defined by increased secretion of adrenal androgens but coincides approximately with the eruption of the first permanent molars and the behavioral and cognitive changes known as the 5- to 7-year shift (15, 16). Gonadarche marks the start of the transition from life as a non-reproductive consumer to life as a reproductive provider. Sexual maturation may be associated with dispersal from the natal group.

The question whether to invest in a child comes logically before the question how much to invest. The next section considers "decisions" to terminate investment and redirect resources to other fitness-enhancing activities. Subsequent sections discuss genetic conflicts over amounts transferred and the timing of ontogenetic transitions. These sections will consider 4 disorders of imprinted gene expression (Table 1): Beckwith–Wiedemann syndrome

(BWS) is associated with excess expression of paternal alleles or deficient expression of maternal alleles, and is expected to exaggerate phenotypic effects that enhance a child's patrilineal inclusive fitness at a cost to its matrilineal inclusive fitness. Silver–Russell syndrome (SRS), Prader–Willi syndrome (PWS), and Temple syndrome (TS) are associated with excess expression of maternal alleles, or deficient expression of paternal alleles, and are expected to exaggerate phenotypic effects that enhance a child's matrilineal inclusive fitness at a cost to its patrilineal inclusive fitness.

Selective Abortion and Infanticide. The death of a child (or embryo) is associated with a fitness cost for its parents and an inclusive fitness cost for other kin, but this loss of fitness via the child can sometimes be compensated by fitness gains via other individuals. Each individual will favor termination of investment in a child if resources can be redeployed to other uses that yield a higher return of inclusive fitness. The costs and benefits of termination may be weighed differently by different group members, depending on their relative relatedness to the discarded child and to other individuals who would benefit from its abandonment. The child is the group member most closely related to itself and, therefore, the least likely to favor its own elimination.

If a child were to die at 7 years, it were better that an infant die at 7 months; if an infant were to die at 7 months, it were better that a babe die at 7 days; and if a babe were to die at 7 days post partum, it were better still that an embryo die at 7 days post conception. It takes 1 death to eliminate 1 copy of a deleterious dominant allele (or 2 copies of a deleterious recessive allele), and the death will have less effect on parental fitness the earlier it occurs. Reproductive compensation for early deaths thus favors the evolution of screening processes, operating before major commitment of resources, that convert small differences of expected fitness into lethal differences (17–19).

Early embryos are easily replaced. Therefore, mothers are expected to be "fastidious" about which embryos implant, and to abandon embryos much more readily than they would abandon a child. The genetic diseases we see at birth are disorders that were compatible with prenatal survival and that evaded detection in utero. Two considerations probably contribute to the inefficiency of prenatal screening. The first is the difficulty of testing many aspects of postnatal gene function in embryos. The second is parent–offspring conflict: embryos have less stringent criteria for continuation of pregnancy than mothers (19).

Mothers probably have effective control over whether, and how much, to invest in offspring during the earliest stages of pregnancy. Implantation, however, marks a major shift in power from mother to offspring. Once an embryo is securely ensconced within the uterus, the offspring has greater control over the delivery of maternal investment than it has at any postnatal stage. Beyond the early stages of pregnancy, a fetus probably has an effective veto on termination of maternal investment and effective control over when to be delivered.

Power to control maternal investment shifts decisively back toward the mother at birth, when a nipple replaces the placenta as the conduit for nutrient transfer. The immediate postnatal period may be the first opportunity to abandon an offspring since the earliest stages of pregnancy (20, 21). Decisions to abandon infants may be influenced by group members with genetic interests distinct from either mother or infant.

Till Birth Do Us Part. Genes of paternal origin are predicted to promote increased demands on mothers during pregnancy whereas genes of maternal origin are predicted to promote reduced demands (6, 7). Strong support for these predictions comes from BWS and SRS, the former associated with fetal overgrowth and the latter with intrauterine growth retardation (22, 23).

The notoriously tight fit between the size of the fetal head and the width of the maternal pelvis suggests that fetuses "choose" to remain inside their mother until the last practical moment. Offspring must have been substantially safer within the uterus than at the breast if the benefits of prolonged gestation were to have outweighed the increased risks of birth complications. I conjecture that longer gestation enhanced the average fitness of offspring but reduced the average fitness of their mothers. There is limited evidence for effects of imprinted genes on gestation length. Gestation is shortened by 2–3 weeks in SRS (22) and preterm and postterm delivery are both increased in PWS (24).

Infancy and Early Childhood. The costs of lactation are borne directly by mothers, although maternal costs may be subsidized by other group members. Supplemental foods are typically introduced early, usually by 6 months (25). At first, these foods must be specially prepared because the infant's gut and dentition are immature. In natural fertility populations, cessation of suckling usually occurs at some time before a child's third birthday with the onset of the mother's next pregnancy (26). More intense suckling and later introduction of solid food prolong lactational amenorrhea (27). Conversely, less intense suckling would shorten interbirth intervals and cause earlier weaning.

Shorter interbirth intervals are associated with increased maternal fecundity but reduced offspring survival (28). Therefore, maternally-derived alleles of infants are predicted to favor lower intensity suckling, greater appetite for supplemental foods, and earlier weaning than paternally-derived alleles. Poor suck is characteristic of infants with SRS, PWS, and TS (24, 29–32). Moreover, infants with SRS show little interest in food and require small frequent feeds (29–31). The large tongues of infants with BWS (33) suggest a role for paternally-expressed genes in the development of the infant's suction pump.

Postnatal feeding is severely perturbed in PWS. This syndrome has been classically described as having 2 phases: poor suck and failure to thrive in infancy, followed by hyperphagia and onset of obesity in early childhood. Recent reviews suggest a more complex 3-phase history with the onset of obesity (18–24 months) occurring before the onset of hyperphagia (5–13 years) (34, 35), but other reviews continue to describe hyperphagia as commencing before excessive weight gain (24).

Two articles have interpreted the change in appetite observed in PWS as a reflection of evolutionary conflict between maternal and paternal alleles over food transfers from parents. Haig and Wharton (36) proposed that paternally-expressed transcripts promote suckling during early infancy but inhibit appetite for supplemental foods at the time of weaning. In the absence of these transcripts, infants with PWS have poor suck during the period of exclusive lactation but develop an insatiable appetite at the time of weaning. The effect of these transcripts was to increase reproductive costs to mothers by extending lactational amenorrhea for the benefit of the offspring (36). Úbeda (37) argued that fathers pay a greater proportion of provisioning costs after weaning and that maternally-expressed transcripts from the PWS region increase demands on fathers for the benefit of weaned offspring. Both articles were based on a 2-phase model of PWS with onset of hyperphagia at the time of weaning. If the onset of hyperphagia is delayed to 5 years or later, then these models may need to be revised because this age correlates more with adrenarche than weaning.

Adrenarche and Preadolescence. Adrenarche occurs at about the age (5–7 years) that the child's immediately younger sib is being weaned because the child's mother is pregnant with the next younger sib. This is an age of significant behavioral and cognitive changes. Children are given more responsibilities, interact more with peers, and develop social norms of reciprocity (15). The sharing decisions of 3- to 4-year-olds are mainly self-centered, whereas 7- to 8-year-olds will share food equitably within their

social group (38). Whether these changes are influenced by adrenal androgens is currently unknown.

I conjecture that adrenarche occurs at an age when the child's sustenance was shifting from predominant reliance on the household pot to greater reliance on the communal pot and self-provisioning. Therefore, genes of maternal origin might be expected to favor earlier adrenarche than genes of paternal origin. Consistent with this hypothesis, premature adrenarche is common in PWS (39), but I know of no data that allow comparison of the relative timing of adrenarche and onset of hyperphagia in PWS.

Children older than 6 years are often expected to work and, cross-culturally, women rather than men are the principal beneficiaries of children's labor (40). Anthropologists have observed marked variation in children's contribution to their own upkeep among hunter-gatherer societies (41, 42). The hyperphagic phase of PWS is associated with behaviors that have been variously described as "foraging" and "food-stealing" (43). It is possible that these terms encompass functionally distinct behaviors. Foraging could reduce withdrawals from the household pot, whereas stealing could increase withdrawals from the communal pot, with both behaviors benefiting genes of maternal origin, but this is mere speculation until the behaviors are better characterized.

Postnatal growth in BWS is characterized by marked acceleration of bone age in infancy and early childhood. Final height is on average 2.5 SD above the mean (44). By contrast, bone age is often delayed in young children with SRS and final height is on average 3.6 SD below the mean (45). This reduction in final height results from slow growth in utero and during the first postnatal months with absence of subsequent catch-up growth (22, 46). In both BWS and SRS, spontaneous puberty occurs at the normal age (44, 45). Length in PWS is within the normal range through the first postnatal year, but declines to the third centile by 3 years, with a further loss in relative height due to absence of the pubertal growth spurt (46).

Fetal and early infant growth are severely perturbed in BWS and SRS. Therefore, genes from chromosome 11p15.5 appear to be major regulators of growth during this period. By contrast, birthweight is within the normal range in PWS, but growth subsequently falters. Therefore, paternally-expressed genes from chromosome 15q11-q13 appear to promote postnatal growth, at least in part, via increased secretion of growth hormone (GH) because GH therapy restores normal adult height in individuals with PWS (47). These observations suggest 2, partially dissociable, phases of growth during early childhood (48).

Childhood is a period of prolonged slow growth. Clinical data from imprinting disorders suggest paternally-expressed genes promote, and maternally-expressed genes inhibit, childhood growth. This implies that larger size benefited offspring at a cost to their mothers' residual reproductive value, although the nature of this tradeoff is not altogether clear. Slow growth, with delayed puberty, would have reduced the rate at which food needed to be supplied to offspring but could have increased the total transfers needed to raise a child to independence.

Puberty. Premature puberty is characteristic of TS (49). Precocious early signs of puberty are also common in PWS (50), but pubertal progression is incomplete, with a weak or absent growth spurt (24, 51). Thus, imprinted genes influence the timing of gonadarche and the pubertal growth spurt, but a clear pattern is absent, perhaps because of the complexity of the underlying selective forces.

In my simple models, gonadarche occurs in the natal group of both sexes. But, when a young couple reproduces, their offspring consume resources in his natal group but her dispersal group. The selective forces associated with the onset of gonadarche are complex because they depend on the relative timing of the transition from being a net consumer to a net producer, of the dispersal of females, and of first reproduction in both sexes.

Offspring presumably benefited from each additional year of prereproductive development by accumulating social, and other, experience that allowed them to function as more effective adults, but this learning period was subsidized by withdrawals from the household and communal pots. From the perspective of other group members, ecological conditions could shift the balance between the benefits of adding an extra adult-sized contributor to the communal pot (favoring earlier male entry to adulthood) and the costs of increased competition from adding an extra household (favoring delayed male entry to adulthood). In contrast, if a daughter's extended sojourn in her natal group increases local resource competition, then one might predict that her paternally-derived genes would favor earlier maturation and dispersal.

Age at puberty is variable among and within human groups and shows strong secular trends (52). Moreover, pubertal timing appears sensitive to both nutritional and social cues. For example, children born in the developing world, but adopted by European families, have high rates of precocious puberty (53) and father absence is associated with early menarche (54). Whether this variability reflects evolved responses to cues of local relatedness and resource transfers remains an open question.

Evolution of Human Life History

Humans take longer to reach nutritional independence than other great apes but have shorter interbirth intervals (25, 55, 56). As a consequence of these two derived features of human life history, mothers often care for "litters" of different-aged offspring. Roughly speaking, a human mother can produce 2 offspring in the time it takes a chimpanzee mother to produce one. Weaning, adrenarche, and first molar eruption are approximately contemporaneous in chimpanzees, but a human mother is weaning her second offspring by the time her first offspring is undergoing adrenarche and cutting its first molar. As a further contrast, a chimpanzee weanling is responsible for feeding itself but a human weanling is fed by others for many years.

I conjecture that prolonged maturation was an adaptation of human offspring that enhanced their individual fitness at a cost to their mothers' fecundity whereas early weaning was an adaptation of mothers that enhanced their fecundity at the expense of offspring survival. This hypothesis is based on substantial evidence that paternally-expressed genes favor more intense suckling, and suggestive evidence that maternally-expressed genes favor earlier sexual maturation. The observation that imprinted genes influence resource transfers and ontogenetic transitions suggests that our distinctive life history has been shaped by conflicting interests of different sets of genes distributed among the individuals of social groups. The resulting life history may be an evolutionary compromise with substantial inefficiencies because of conflict costs.

One of the most promising avenues for testing these ideas will be detailed longitudinal studies of feeding behavior, adrenarche, and pubertal progression in children with various imprinting disorders. Such studies would not only be of evolutionary interest but also of clinical value. For example, many of the health problems in individuals with PWS are associated with obesity and hyperphagia, but the ages of onset of excessive weight gain and excessive feeding appear to be highly variable among individuals. It would be useful to know whether this variability is "random noise" or is systematically associated with differences in family dynamics and how food is presented.

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